Naval Medical Research Institute
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THE EFFECT OF TEMPERATURE ON DECOMPRESSION AND DECOMPRESSION SICKNESS RISK: A CRITICAL REVIEW

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There are long-held beliefs regarding temperature effects on dive outcome. One accepted tenet is that decompression sickness (DCS) risk increases during exposures to cold water. It is also commonly held that post-dive hot water showers encourage the onset of DCS. The question of thermal effects on DCS was raised most recently in response to observations of DCS cases after the introduction of hot water suits during the salvage effort for TWA Flight 800. We conducted a literature review using 4 biomedical reference databases to locate human and animal studies associated with diving, caisson work, and aviation. Studies were selected for inclusion if they examined the relationship between thermal conditions and DCS risk, the production of venous gas embolism, or inert gas exchange. We conducted a second search for the effects of showering on DCS and examined the Naval Medical Research Institute (NMRI) DCS database for cases of DCS associated with showering. Accepted epidemiological criteria for the evaluation of causal relationships were applied to the studies we found on the subject.

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EXECUTIVE SUMMARY

There are long-held beliefs regarding temperature effects on dive outcome. One accepted tenet is that decompression sickness (DCS) risk increases during exposures to cold water. It is also commonly held that post-dive hot water showers encourage the onset of DCS. The question of thermal effects on DCS was raised most recently in response to observations of DCS cases after the introduction of hot water suits during the salvage effort for TWA Flight 800. We conducted a literature review using 4 biomedical reference databases to locate human and animal studies associated with diving, caisson work, and aviation. Studies were selected for inclusion if they examined the relationship between thermal conditions and DCS risk, the production of venous gas embolism, or inert gas exchange. We conducted a second search for the effects of showering on DCS and examined the Naval Medical Research Institute (NMRI) DCS database for cases of DCS associated with showering. Accepted epidemiological criteria for the evaluation of causal relationships were applied to the studies we found on the subject.

Seventeen textbook articles, reviews, and symposia pertaining to the relationship between thermal factors and DCS risk were examined. Twenty-three original human studies were reviewed, 13 of which met our inclusion criteria. Five animal studies were reviewed but are not included in this report because they were not thought to contribute to the questions of interest.

We found no definitive study that demonstrates a causal relationship between thermal conditions and DCS risk or the magnitude of any effect. There is sufficient evidence to believe that the studies in the aggregate weakly support the hypothesis that DCS risk is increased when a diver is warm on the bottom, cold during decompression, and cold on the surface. One study suggests that the use of hot water suits at the same temperature on the bottom and during decompression is likely to increase DCS risk. The studies are insufficient to determine if being cold on the bottom and cold during decompression will increase DCS risk. The magnitude of any thermal effect cannot be precisely determined from these studies, but it is probably small. The hypothesized physiological mechanisms responsible for the effect include changes in inert gas solubility and blood flow in peripheral tissue.

Based on the conclusions of this review, it would seem prudent to keep divers relatively cold on the bottom and relatively warm during both in-water and surface decompression. It is

not clear from existing studies if providing divers with heated gas would be an advantage, since the limited evidence that does exist suggests the effect is influenced by skin tissue temperature. One author suggested that divers should be warmed after a dive in a hot bath. While this may be impractical, keeping divers warm on the surface, particularly in recompression chambers during surface decompression, seems reasonable. At present there is no practical way to monitor physiological changes in an operational setting that would enable us to predict whether a diver's temperature would increase his risk of developing DCS. Additional decompression time can always be added to account for any increased DCS risk, particularly in surface decompression procedures using oxygen, and this may be the practical procedural approach. There is no definitive evidence that hot showers after a dive precipitate DCS.

Further analysis of existing thermal and DCS data might be fruitful in strengthening our confidence in a causal relationship. A precise estimate of the magnitude of any thermal effect would require a major human dive trial. Examination of hypothesized mechanisms of action might provide insight into the broader question of DCS pathophysiology. Research should begin by examining the data collected from the salvage of TWA Flight 800.

INTRODUCTION

It is well established that the major determinant of decompression sickness (DCS) risk is the dive profile. The extent to which other environmental factors, such as temperature, impact a diver's risk for DCS is not as well characterized. There are long-held beliefs regarding temperature effects on dive outcome. One accepted tenet is that DCS risk increases during exposures to cold water. Navy diving procedures require divers to be shifted to a longer decompression schedule than would normally be used, whenever divers are "exceptionally cold during the dive."[50] The presumption is that cold water conditions predispose the diver to an increased risk for DCS, and an allowance is necessary to decrease the diver's risk of DCS through dive table selection. It is also commonly held that post-dive hot water showers may encourage the onset of DCS. Some US Navy diving supervisors limit or strictly control post-dive hot showers.

The question of thermal effects on DCS was reopened in response to observations of DCS cases during the salvage effort of TWA Flight 800. The DCS cases appeared to cluster after divers switched from wetsuits to free-flooding hot water suits. This experience seemed to contradict the standard Navy doctrine described above. While the data from the salvage operation have yet to be examined to determine if the apparent clustering of DCS cases was related to the introduction of hot water suits, the Naval Sea Systems Command requested that the Naval Medical Research Institute (NMRI) review the literature for evidence that diver body temperature significantly affects the decompression process to the extent it could increase or decrease the occurrence of DCS in an otherwise constant environment.[49] We expanded our review to include ambient temperature effects as well as diver body temperature. This paper reports on the findings of that review.

METHODS

Literature Review

To establish a causal relationship it is necessary to examine human studies which compare an outcome measure, in this case DCS, between groups exposed and unexposed to the hypothesized causal factor, in this case thermal conditions. Research in animals can be used to

examine mechanisms of action and support observations in human studies. We first examined diving and aviation medicine textbooks and symposia to understand the current thinking in the field. We then conducted a search of multiple databases (National Library of Medicine's MEDLINE, Defense Technical Information Center, Federal Research In Progress, and the Undersea and Hyperbaric Medical Society Library) to locate original human and animal studies which relate the effects of temperature, environmental or otherwise, on the incidence and mechanisms of DCS associated with diving, caisson work, and aviation. Studies were selected for inclusion if they examined the relationship between thermal conditions and DCS, the production of Doppler-detected venous gas emboli (VGE), or inert gas exchange. We consider studies that report on DCS as the outcome measure to be of the greatest relevance, while other outcome measures are only supportive since the relationships between VGE, inert gas exchange, and DCS are imprecisely defined and controversial. We also conducted a search for the effects of post-dive showering on DCS incidence. In addition to the other literature databases, we searched the NMRI decompression database, which contains military research laboratory diving records of the United Kingdom, Canada and the US, between 1945 and the present, to locate cases of DCS associated with showering.

Analysis

Determining whether or not a causal link exists between two associated factors is a common problem in epidemiology. The "gold standard" for determining whether or not a relationship is causal is a large, randomized, blinded trial in which the exposure of interest, in this case temperature, is applied to a group of individuals in a controlled manner and the effect of the exposure is observed. Since this type of study is often difficult to conduct, especially for environmental or occupational exposures, epidemiologists have developed criteria for assessing causality from information available in other types of studies. These criteria were first devised by Hill,[32] applied by the US Surgeon General to the problem of smoking and lung cancer,[47,48] and generally adopted in epidemiology after some debate and modification[30,41] to provide a useful framework to help establish causality of an association. The criteria we use are those most recently outlined by Goldsmith[30] and include: strength of the association, control of confounding

and bias, consistency of findings, dose-response relationship, temporal cogency, specificity, biological plausibility, and overall coherence. The nature of each criterion is described below. These criteria were applied to the original studies that we uncovered in our review.

Strength of Association

Strong associations, commonly defined by epidemiologists as an incidence rate ratio between an exposed and unexposed population in an observational study of *at least* 2.0 (equivalent to a 100% increase in incidence),[30,57,58] are more likely to be causal than weak ones. This is not to suggest that only effects which cause doubling of incidence are important, but rather that effects less than this magnitude can easily be caused by confounding or bias. If an association were in fact due to confounding or bias then the erroneous factor would have to be strong, and would presumably not remain undetected for very long. Conversely, unknown confounders would likely be associated with very small rate ratios. The size of a study indirectly influences the strength criterion because the larger the study the more confident we can be that the observed rate ratio is not due to chance alone. However, a large study alone does not assure that the observed rate ratio accurately measures the true relationship between two associated factors. This can only be assured by control of confounding and bias through careful study design.

Control of Confounding and Bias

This criterion must be considered when interpreting strength of association. Studies with a weak association and identifiable confounding and bias bring into question any claim to causality. A **confounding factor** is one which is related to both the outcome measure and the factor under study, and which may have an equivalent or stronger role in causation than the factor under consideration. The biggest known confounder for determining a causal relationship between thermal factors and DCS is the dive profile, but there are other potential confounders such as immersion and exercise. A **bias** is a systematic error in collecting or categorizing data. If the manner in which information is obtained or if the subjects in the groups being compared are different, and these differences are related to the factor being studied (*e.g.,* thermal condition)

then the resulting bias could lead to an incorrect conclusion about the exposure-outcome relationship. Many types of bias have been identified and specific biases are noted in each study analysis.

Consistency of Findings

Studies in which different investigators apply a variety of study methods yet which result in the same or very similar findings lend strength to the causal link. Hill specified that for this criterion to be met, a consistent effect must be found in different populations, at different times, and by different persons.[32]

Dose-Response Relationship

This criterion requires the demonstration of a relationship between the amount of exposure and the severity or incidence of a disease. For example, if cold dives actually increase DCS risk then the incidence of DCS should decrease for dives in warmer water (all other conditions being equal). Similarly, warm water dives should result in fewer cases of severe DCS.

Temporal Cogency

This criterion simply requires that the proposed cause precede the measured effect. Rothman points out that perhaps only temporality is a necessary condition for causality; even a "part of the concept of causality."[41] To meet this criterion, the subjects must have undergone a decompression and thermal exposure prior to measuring one of the three outcomes of interest (DCS incidence, VGE, or inert gas elimination).

Specificity

This criterion requires that a one-to-one relationship exist between the exposure and disease. This condition would be met if a specific thermal exposure resulted in a unique manifestation of decompression sickness. There is currently no known unique manifestation of DCS due to thermal effects alone. Moreover, this condition is not met by DCS in general because there is not a one-to-one relationship between diving and DCS symptoms and signs.

This is because DCS symptoms and signs are similar to those caused by other conditions such as musculoskeletal injury or numbness after a cold dive.

Biological Plausibility

This criterion is based on the premise that effects demonstrated to be biologically plausible are more likely to be causal. This criterion is met if a study demonstrates an association between a hypothesized mechanism of action of thermal conditions and one of the three outcome measures of interest (DCS incidence, VGE, or inert gas elimination).

Coherence

"The cause-and-effect interpretation of our data should not seriously conflict with the generally known facts of the natural history and biology of the disease."[32] We interpret this to mean that the criterion of coherence is not maintained if any known facts about decompression sickness are undermined by any of the studies we evaluated.

RESULTS

Literature Review

We found the belief that thermal conditions, specifically cold and the use of hot-water suits, influence DCS incidence pervades diving and aviation medicine textbooks, panel discussions, and review articles.[25-29,31,33-38,42-44,46,52-53] The most widely accepted framework for thinking about the relationship between thermal factors and DCS, in these sources, is that thermal effects are different depending on the phase of the dive. It is believed that warm conditions on the bottom favor increased inert gas uptake and increased DCS risk, and cold conditions during both in-water and surface decompression slow inert gas elimination and thereby increase DCS risk. In the context of the different phases of a dive, the US Navy diving procedures appear based on the assumption that cold conditions during decompression will outweigh any beneficial effect from cold conditions on the bottom. Further, an increased risk of DCS after hot-water suit diving assumes that warm conditions on the bottom outweigh any beneficial effects from warm

conditions during decompression. Given this framework, our review focused on uncovering original studies which might support these beliefs.

Despite the generally held belief that temperature affects a diver's DCS risk, there have been relatively few attempts to evaluate this association. Of the 23 original human studies that we reviewed,[1,2,4-7,10-13,15-24,26,39,45] thirteen met our inclusion criteria: 5 hyperbaric studies with DCS as the outcome measure,[4,11,12,16,17] two hyperbaric studies with Doppler-detected VGE as the outcome measure,[5,10] one hypobaric-immersion study with DCS as the outcome measure,[2] and five aviation DCS studies.[1,6,7,13,15] Five animal studies were found,[3,8,9,14,40] and four of these were reviewed.[3,8,9,14] The animal studies gave inconsistent results and had numerous methodological problems that are beyond the scope of this review. We consequently decided to limit our presentation to human studies. The 13 human studies are summarized and analyzed in this review. None of the human studies examined the three phases of the dive independently. Most studies typically combined bottom-time and pre-surface decompression as one set of thermal conditions, and defined surface conditions as another. We grouped studies that examined the same phases of the dive.

No studies were found which specifically examined the effect of showers on DCS risk, although one study[10] reports a possible relationship through a serendipitous observation. We also were apprised of a large human study where post-dive showering was standard practice (Weathersby, personal communication), and where the overall DCS incidence was surprisingly low.[55] In the NMRI database, we reviewed 393 DCS cases and 546 marginal cases. (Marginal cases are defined as cases which had symptoms and signs thought to be decompression-related but that did not require recompression treatment.) Of the cases reviewed, we found 5 DCS cases and 1 marginal case in which the onset of DCS was associated with showering. We incidentally noted two cases of DCS in which mild symptoms improved after a hot bath.

Study Analysis

Human hyperbaric studies which examine <u>combined bottom and pre-surface decompression</u> effects on DCS risk

Weathersby[17] (1990)

Weathersby compared DCS risk between 797 *dry* dives and two groups of *wet* dives (244 and 483) conducted in military hyperbaric laboratories in the US and Canada, using a variety of DCS risk models to account for differences in dive profiles. The dry dives were generally resting and thermally comfortable while the wet dives were generally working and cold.

The relative risk of DCS in wet dives as compared to dry dives was determined to be from 0.8 to 1.14 with an upper 95% confidence limit of 1.8. However, this study is confounded by immersion, exercise, and different thermal conditions. The study controls for dive profile by using a DCS risk model with parameters estimated from the data. If it is assumed that the increase in relative risk on wet dives is due to colder temperature, the results of this study are consistent with the hypothesis that cold dives increase DCS risk on which current Navy procedure is based. The study does not examine biological plausibility or a dose-response relationship. However, it is temporally cogent and coherent.

Shields[12] (1986)

Shields and Lee were contracted in 1986 as an independent investigatory unit to evaluate the safety of the US Navy surface decompression tables as used in North Sea commercial diving. Anecdotal evidence had suggested that surface decompression tables and the use of hot water suits were responsible for an increased incidence of DCS, notably neurological (Type II) symptoms. They performed a retrospective analysis of air dives conducted offshore in the United Kingdom sector of the North Sea over a two-year period (1982 and 1983) for the incidence of DCS and the contribution of influencing factors, such as thermal protection.

Their analysis surveyed 25,740 individual dive records. Seventy-nine cases of DCS (0.31%) were recorded: 44 cases of Type I and 35 cases of Type II DCS. To evaluate the data, the investigators derived an index of decompression stress they referred to as the Decompression Penalty Index (DPI). Real-time recording of dive profiles was not conducted.

They found for dives with the most severe decompression stress (DPI > 60) that DCS incidence was 2.4% (16/680) for divers in hot suits, but only 0.5% (2/442) for divers wearing passive thermal protection. For less severe dives (DPI < 60) the DCS incidence was similar (0.4 and 0.3%, respectively). They also noted that 50% of cases were Type II (31/62) for divers wearing hot suits, but only 38% (3/8) for divers with passive thermal protection. They concluded that the major factor involved in the occurrence of DCS cases is the severity of the hyperbaric exposure of the dive, with the use of hot-water suits acting as a contributory factor both to the overall incidence of DCS and to the proportion of Type II cases.

Using data from this study we calculated the DCS incidence ratio for the most severe dives was 4.8 (0.024/0.005) for divers in hot suits compared to divers wearing passive thermal protection. For less severe dives the DCS incidence ratio was 1.3 (0.004/0.003). We also calculated that the ratio of the proportions of Type II to Type I cases was 1.3 (0.50/0.38) for divers wearing hot suits compared to divers with passive thermal protection. The greatest potential confounder to this study is the dive profile, because they were not recorded in real time. Another potential confounder is the use of their severity index, which may not accurately measure decompression stress.[54] Moreover, the average DPI score for the hot water suit divers and the passive thermal protection group may not be the same even though both are greater than 60 (*e.g.*, hotwater suit DPI could be 75 while passive thermal protection divers DPI could be 61). If this was the case, the greater decompression stress alone could account for the observed difference. The authors note their uncertainty in collecting data on all dives, and even all cases of DCS. They conducted no statistical analysis because of the small number of cases, relying on trends in the data to make their conclusion. The results of their analysis are consistent with the hypothesis that warm on the bottom increases DCS risk. However, the study does not examine biological plausibility or a dose-response relationship. The study displays temporal cogency and is coherent.

Van Der Aue[16] (1951)

This report covers the development of air dive surface decompression procedures using oxygen at the Naval Experimental Dive Unit. While not expressly the subject of the study, the influence of water temperature on DCS risk was commented on by the authors. Four divers on

the same diving schedules in warm (74 and 75 °F) and cold (40 to 50 °F) water developed DCS in the warm water but not in the cold. The authors felt that "these four instances tend to substantiate the current theory that the rate of gaseous absorption by the body tissues is less in a cold than it is in a warm environment."

Because this information is anecdotal and because temperature was not even a study variable, a strength measure cannot be calculated and confounding and bias do not apply. It is consistent with the warm on bottom increases DCS risk hypothesis, does not examine biological plausibility or dose-response, is temporally cogent, and is coherent.

Human hyperbaric studies which examine post-dive thermal conditions on DCS risk

Broome[4] (1993)

An epidemiological review of British civilian accident records was conducted by Broome to evaluate the influence of air temperature, wind chill, and other external variables on DCS incidence. His study examined whether differences in surface thermal exposures could explain greater than expected proportions of cases, by his categorization of safe and risky dives. Safe dives were arbitrarily defined to be those dives conducted within the no-stop limits of the Royal Navy Air Decompression Table 11 (RN Table 11).

Study cases were selected from all diving accidents reported to the United Kingdom Institute of Naval Medicine over the six year period from 1984-1989. Dive profiles were patient reported. All cases diagnosed as cerebral arterial gas emboli, pulmonary barotrauma, or DCS with neurological manifestations were rejected. Non-diving related illness and cases unresponsive to recompression treatment were also rejected. Weather data were collected from archived meteorological records. Of the 831 diving accident records reviewed, 177 cases of DCS were identified as meeting his inclusion criteria: 116 were single dives (36 *safe*, 80 *risky*) and 61 were repetitive dives (6 *safe*, 55 *risky*) for totals of 42 *safe* and 135 *risky* profiles.

Broome assumed that the degree of risk of the dive profile was selected independently of the air temperature. Because of this assumption one would expect the proportion of risky dives occurring in warm air to be the same as the proportion of safe dives occurring in warm air. If air temperature has no effect on DCS then the proportion of DCS cases should be directly related to

the proportion of risky and safe dives made, and not on the air temperature. Therefore, the ratio of DCS cases on risky and safe dives should remain constant across air temperature.

No DCS cases were associated with *safe* dives when the air temperature was greater than 18.5°C, and fifty percent of cases in safe exposures occurred with an air temperature below 13°C. Twenty eight per cent of all DCS cases associated with *risky* dives occurred with air temperature above 18.5°C, while 25% of *risky* dive associated cases occurred below 13°C. This data suggests that air temperature affects the ratio of the proportion of cases in each air temperature group, contrary to expectation assuming no air temperature effect. Moreover, if the air temperature were more than 1.6°C warmer than the water temperature, only 22% of DCS cases associated with *safe* dives occurred compared with 50% of DCS cases associated with *risky* dives. A larger percentage of DCS cases on safe exposures (45% versus 25% for risky dives) were associated with a high wind-chill index.

There are several limitations in this study, notably the lack of information on the total number of dives conducted in both the safe and risky groups. This precludes the calculation of incidence and rate ratios. Moreover, there are several confounders and biases in the study. The most prominent confounder is the self-reporting of dive profiles by the patients themselves which is known to be inaccurate. Misclassification bias may be introduced by the arbitrary choice of RN Table 11 no-decompression dives as safe. One analysis of RN decompression tables suggests that DCS risk is approximately the same and even decreases for some decompression dives when compared with no-decompression dives.[54] The assumption that selection of the dive profile is independent of weather conditions may also be incorrect. For example, divers may select short no-decompression dives if surface conditions are poor. Water temperature was also different for the safe and risky groups. Selection bias was incurred by accepting only Type I DCS cases. This approach assumes that thermal effects act preferentially on Type I DCS cases only. Selection bias was further incurred by accepting only those responsive to recompression treatment, even though DCS is a clinical diagnosis and actual DCS cases may have been rejected. The results are consistent with cold surface conditions being conducive to DCS. This study does not examine biological plausibility or a dose-response relationship, but it is temporally cogent and coherent with existing literature.

Paton and Walder[11] (1954)

The Medical Research Council of the United Kingdom investigated the incidence of compressed air illness in caisson workers during the construction of the Tyne Tunnel between 1948-50. They attempted to find conditions which influenced the observed DCS rate of 0.87% (350/40,000), including weather conditions. They examined weather conditions because of the widespread belief among workers that damp and "cold, frosty weather" caused an increase in susceptibility to DCS. The study compared meteorological data for conditions 5 miles from the construction site to DCS incidence, over weekly intervals. They concluded that no correlation could be found between DCS rate and any of the weekly averages for maximum or minimum temperature, or any other climactic factor, but do not report incidence by temperature. The study is confounded by the fact that the temperature in the tunnel was not reported, the decompression chamber was heated in non-summer months, and workers waited in a heated room for several hours after reaching the surface. In addition the authors mentioned the possibility of an acclimatization effect and elimination of workers prone to DCS, but did not control for these factors in their analysis of temperature effects. This study is neither consistent with the thermal effect hypothesis, nor does it examine biological plausibility or dose-response. It fails to control adequately for confounders and bias. It is temporally cogent and is coherent with existing literature.

Human hyperbaric studies with Doppler-detected venous gas emboli as the outcome and post-surface decompression effects

Mekjavic[10] (1989)

Mekjavic and Kakitsuba set out to demonstrate the effect of *peripheral* temperature on detected venous gas bubbles, while maintaining stable and normothermic core temperatures. Four divers were subjected to two separate air chamber dives at a constant 25°C under simulated near-saturation conditions (12 hours at 30 fsw (1.9 ATA)), and subsequent dropouts to the surface (1.0 ATA). Subjects were then observed over 3 hours in temperature-controlled

chambers in one of two conditions (40°C and 10°C) and monitored for precordial venous gas bubble levels, both at rest and during moderate exercise.

Precordial measurements revealed gas bubbles in only one subject during the rest phase (10°C exposure). In the exercising subjects, one subject at 40°C had detectable bubbles, but three of the four subjects in the 10°C exposure had significantly high bubble scores. No symptoms of DCS were apparent at any time during the observation period, however 3 of the 4 subjects reported DCS symptoms (pruritus and mild shoulder pain) during a hot shower taken after the end of the observational period on the 10°C dive, compared with 0 of 4 on the 40°C dive. This high incidence of DCS symptoms terminated the study.

A rate ratio from thermal effects prior to the introduction of showering cannot be calculated because DCS only occurred after showering. A strength of this study lies in their control for confounding variables, through the regulation of subject's diet, activity and dive protocol. Unfortunately this study observed a very small number of divers (N = 4). VGE and DCS outcomes are not blinded to thermal exposure causing a potential bias. It is consistent with warm being better during decompression, supports biological plausibility, does not examine a dose-response effect and is temporally cogent and coherent.

Human hyperbaric studies with Doppler-detected venous gas emboli as the outcome and pre- and post-surface effects

Dunford and Hayward[5] (1981)

Dunford and Hayward conducted a diving experiment in the Puget Sound to evaluate the effect of environmental temperature on post-dive VGE. Ten male subjects dove to a depth of 78 fsw (3.36 ATA) at a constant water temperature of 10°C. The divers were configured in one of two thermal protective garments to achieve warm and cold conditions: warm (W), insulated in variable-volume dry suits and insulated undergarments, and cold (C) utilizing 1/8 inch wet suits. Following the dive each subject was either "actively rewarmed" in a warm bath (B), over a programmed temperature range (27°–42°C) or "passively rewarmed" in an insulated sleeping bag (I). The subjects were sequence randomized for participation in each of the four possible

protocols (WI, WB, CI, CB) for a total of 40 dive exposures. During the post-dive rewarming phase, precordial VGE were assessed at 20 minute intervals over a 140-minute period.

The average diver bottom time was 40.8 minutes, which for a 78 FSW dive is on the cusp of the US Navy Dive No-decompression limit (80 feet for 40 minutes). There were no observed signs or symptoms of DCS. The VGE scores revealed levels in the warm dives that were initially three times higher than the cold dives. Warm insulated dives (WI) had higher scores than cold insulated (CI) dives ($P<0.05$). Warm dives with post-dive bath (WB) were also significantly higher than the cold dives with bath (CB) over most of the Doppler measured time periods ($P<0.05$) (notably, not the later three time periods). No significant differences were noted between the two cold dives (CI, CB).

Dunford and Hayward concluded that VGE counts for warm dives are greater than for cold, irrespective of the post-dive thermal condition imposed. The use of VGE scores as a precursory marker for DCS symptoms is controversial. However, if viewed as an indicator for inert gas load, then this study would support directly the hypothesis that cold during the compression phase, and less strongly that warm on the decompressive phase, would reduce the amount of inert gas uptake. A source of confounding lies in comparing the effects of a *dry* insulation barrier to *immersion* in a bath during the decompression phase, as this ignores any differences in gas exchange between air-skin and water-skin boundaries. Because no case of DCS was reported in this study the application of Hill's criteria is limited. It is consistent with warm during decompression being beneficial, the study is temporally cogent, biologically plausible, and coherent. Again however, there is a lack of subject blinding to VGE detection causing a potential bias and it does not examine a dose-response relationship.

Human hypobaric studies with DCS as the outcome measure

Balldin[2] (1973)

The intent of this study was to investigate whether immersion in water under warm conditions would reduce the risk of decompression sickness after a hypobaric exposure. The studies were conducted as hypobaric chamber dropouts from normal atmospheric pressure (1.0 ATA) to a subatmospheric pressure, simulating an altitude of 38,000 feet (155 mm Hg or 0.2

ATA). Ten subjects were exposed to two separate ambient conditions at atmosphere pressure (1 ATA) while 'denitrogenated,' by breathing 100% oxygen. Each denitrogenation exposure was conducted for 25 minutes with the *warm-immersed* subjects immersed to their necks in warm water at 37-37.5°C and the *cold-dry* subjects exposed to an ambient temperature of 22-24°C. Following denitrogenation, each 5-member group was then rapidly decompressed to 155 mm Hg (at unspecified temperature conditions). While decompressed the subjects were exercised and observed over a two-hour period for signs of pain-only DCS. Each subject group was sequentially exposed to each of the two thermal conditions.

Nine of the 10 (90%) dry-cold exposures resulted in bends and 2 of 10 (20%) wet-warm exposures resulted in bends. Balldin reports significant findings ($P< 0.01$) to support that oxygen breathing before hypobaric decompression, with the combination of immersion and warm temperature, is protective for DCS as compared with oxygen breathing under colder and dry conditions. An obvious limitation of this hypobaric study to the issue of thermal effects on DCS is its applicability to diving scenarios. While diving and aviation DCS presumably share a common etiology, the distribution of manifestations of the injury, and the greater gas burden in diving do not make these conditions identical. In addition, the subjects are pre-oxygenated as a means to decrease DCS risk which is a procedure commonly found in aviation but not diving operations. The study does not separate effects of immersion and dry from that of thermal states. Another shortcoming is the lack of reporting of post-decompressive subject temperatures. Balldin's conclusion that a desired mechanism for reducing DCS incidence is to keep subjects in a warm environment (immersed or otherwise), during decompression is consistent with the hypothesis that warm during decompression is beneficial. However, both the immersion effects and the uncertain analog of hypobaric data to diving DCS confound their results. A dose-response relationship is not studied. It supports biological plausibility and is coherent.

Aviation DCS Literature

The earliest published data on the effects of external temperature on decompression outcome emerges from aviation medicine literature published during and just after World War II. As operating altitudes increased the operating temperatures dropped (often reaching -50°F,

-45.5°C) and the DCS incidence was found to increase. Aviation researchers hypothesized that factors other than the hypobaric insult alone might be compounding the natural incidence of bends, and the leading culprit was temperature. The aviation studies which resulted from this period are reviewed by Cook[27] and a summary of these studies is provided.

Fraser and Rose[6] of the Canadian military observed subjects in 1943 at simulated altitudes of 35,000 feet, finding more pain-only symptoms in subjects at -10°F (-23.3°C) compared with those subjects at 70°F (21.1°C). Two American investigators, Anthony *et al*,[1] and Griffin *et al*,[7] arrived at similar conclusions: colder subjects manifest a greater incidence of pain-only bends symptoms than warm subjects. In 1946, Smedal *et al*,[13] evaluated aviation students (N = 1731) at three simulated low-level altitude exposures (less than 35,000 feet). Their exposure periods were much shorter than the prior studies (20 minutes versus at least 3 hours) but they reported a significantly higher DCS incidence during *warm* decompressive conditions. This finding was opposite that of their contemporaries. Finally, in 1947 Tobias *et al*,[15] evaluated whether dorsal hand skin temperatures (N = 24) during simulated ascents to 35,000 feet predicted DCS symptoms. He reported that subjects who developed pain-only bends had significantly lower hand temperatures than those who did not develop DCS. In the aggregate, and except for Smedal,[13] these findings appear to confirm that lower temperatures had an adverse effect during decompression, which Cook summarized as "...men kept warm suffered less pain."[27]

A source of bias pointed out by Nims[37] is that the subjects in these aviation studies were invariably exposed to thermal extremes while dressed in protective clothing. As in the hypobaric study by Balldin,[2] the applicability of hypobaric data to diving DCS risk requires some extrapolation. The validity of these early aviation studies is further confounded by their lack of complete data reporting (subject numbers, study conditions, specific temperature exposures, and symptoms) and calculation of rate ratios. They are consistent with the hypothesis that cold during decompression increases DCS risk and are coherent, but do not examine biological plausibility or dose-response.

DISCUSSION

Summary of Analysis

In summary, all the studies reviewed are limited by confounding, bias, or other methodological flaws. These flaws are particularly evident in the studies by Broome,[4] Van Der Aue,[16] and Paton and Walder,[11] and we chose to eliminate them from further consideration. No study examines all three phases of the dive, nor was dose-response data derived in any of the studies. Only the studies of Weathersby[17] and Shields[12] offer estimates of DCS incidence rate ratios (strength) between warm and cold conditions during the bottom and pre-surface phases of the dive. Shields concludes that being warm during these phases of the dive results in a 4.8 fold increased risk of DCS. Weathersby concludes that being wet and cold on these phases of the dive increases DCS risk by no greater than two-fold compared with being dry and relatively warm. These apparently contradictory results could be reconciled by returning to the premise introduced earlier as the most accepted framework for thermal affects, which relates DCS outcomes to different phases of the dive. The Weathersby study supports the hypothesis that being cold on the bottom outweighs any harm from being cold during decompression, while the Shields study supports the hypothesis that being warm on the bottom eliminates any benefit of being warm during decompression. Nevertheless, only the Shields study presents a rate ratio greater than 2, thus meeting the strength criterion. We conclude that the strength reported in the Shields study supports a causal relationship between wearing hot water suits and DCS risk, whereas the Weathersby study fails to meet this criterion for supporting a causal relationship between cold conditions on the bottom and during decompression and DCS.

The studies of Balldin,[2] Dunford and Hayword,[5] Mekjavic,[10] and the aviation DCS literature[1,6,7,13,15] are all consistent in supporting the notion that cold on the bottom and warm during decompression have beneficial effects on decompression. However, either because of their outcome measure or hypobaric nature, these studies cannot be applied directly to the question of DCS risk during diving. Biological plausibility is also supported because the hyperbaric human studies[2,5,10] support a plausible mechanistic hypothesis. All the studies met the requirement of temporality. The criterion of specificity is not supported because there is not a one-to-one relationship between thermal conditions and DCS as was mentioned in the

introduction. A dose-response relationship was not studied, but this neither adds nor detracts from a causal relationship. Finally, coherence with what is known about DCS supports a causal relationship.

We conclude that the studies, in the aggregate, weakly support the hypothesis that DCS risk is increased when a diver is warm on the bottom, cold during decompression, and cold on the surface. One study suggests that wearing hot water suits is likely to increase DCS risk. The studies are insufficient to determine if being cold on the bottom and cold during decompression will increase DCS risk. The magnitude of any thermal effect cannot be determined from these studies. Physiological mechanisms responsible for the effect have not been determined. Two leading hypotheses include changes in inert gas solubility and blood flow in peripheral tissue. This would lead to increased gas uptake when warm, decreased elimination when cold, and easier supersaturation after a pressure drop when cold, leading to bubble formation and DCS.

Operational Implications

Based on the conclusions of this review, it would seem prudent to keep divers relatively cold on the bottom and relatively warm during both in-water and surface decompression. Recommendations regarding the use of hot-water suits are especially challenging since we do not know the temperature ranges that might cause any increase in DCS incidence stemming from their use. From an operational perspective it may not be desirable to keep the diver relatively colder during the bottom phase since this is when functional impairment from the cold is least desirable. However, a more practical approach may be to limit any diver warming on the bottom to that which still allows job performance. Other factors, such as exercise and inert gas type, also may influence both the thermal status of the diver and the DCS risk. It is not clear from existing studies if providing divers with heated breathing gas would be an advantage, since the limited evidence that does exist suggests the effect is influenced by peripheral tissue temperature. One author suggested that divers should be warmed after a dive in a hot bath.[51] While this may be impractical, keeping divers warm on the surface, particularly in recompression chambers during surface decompression, seems reasonable. At present there is no practical way to monitor physiological changes in an operational setting that would enable us to predict whether a diver's

temperature would increase his risk of developing DCS. Additional decompression time can always be added to account for any increased DCS risk, particularly in surface decompression procedures using oxygen, and this may be the practical procedural approach. However, without additional information the amount of additional decompression needed to account for a given set of thermal conditions cannot be determined *a priori.*

There is no definitive evidence that hot showers after a dive precipitate DCS. Hot showers suppress shivering, heat production, increase evaporative heat loss, and provide little heat transfer and thus do not effectively rewarm.[44] It may be that hot showers adversely affect peripheral blood flow and inert gas solubility as described above, thus increasing DCS risk. However, this has not been studied in the context of DCS. Besides anecdotal reports, only the serendipitous finding in the study by Mekjavic[10] provides the kind of information required. While their findings are suggestive of an association there are three major limitations. First, the diagnosis of DCS was not made by physicians blinded to the conditions of the dive. Second, all of the cases responded to surface oxygen and were not felt to require recompression treatment, bringing the diagnosis of DCS into question. Third, the small numbers do not allow us to conclude with sufficient probability that the events did not occur by chance alone.

Further Research

Additional research could be conducted to strengthen our confidence in a causal link between thermal factors and DCS risk, to better estimate the magnitude of the effect for different phases of the dive, and to recommend modifications to decompression and thermal procedures. A specific goal of such research would be to determine what temperature ranges need to be maintained on each phase of the dive to maximize decompression efficiency. Studies focused on the mechanism of action of such an effect would be useful by providing insight into the fundamental question of DCS pathophysiology and the factors which influence its occurrence.

Observational Studies

Observational studies of large diving operations can potentially add information relating to the thermal question. The salvage of TWA Flight 800 offers the most recent opportunity.

Unfortunately, these studies are not often ideal because of difficulties encountered in collecting the necessary data (*e.g.*, specific dive profiles) and because they usually involve small numbers of DCS cases. U.S. Navy Lieutenant Chris Leffler was an on-site Diving Medical Officer at the TWA salvage who has collected dive and DCS case information from the salvage effort. These data should be reviewed for completeness and analyzed to determine any hot water suit effect on DCS risk. Further refinement in diving data collection and logging should allow for more precise information in any future observational studies.

Existing Databases

We reviewed information in the NMRI database to determine if there was sufficient existing data to help address the questions of thermal effects on DCS. Many of the dives in the NMRI database contain some thermal information (*e.g.*, water temperature). The analysis of wet versus dry dives by Weathersby[17] is based on a portion of the NMRI database. The data discussed from the Van Der Aue report[16] is an example of the type of data that is available for further analysis. The Van Der Aue data (which includes over 1000 dives using surface decompression with oxygen) represents a useful starting point for further study of existing data.

Information concerning another database that was provided from the University of Pennsylvania contains a letter from Dr. C J Lambertsen, and two reports on decompression tables, but no specific data on thermal issues. Dr. Lambertsen states his opinion that a definitive study on the influence of hot water suits on DCS risk had not been performed and describes data (presumably available at the University of Pennsylvania) on Air Surface Decompression O_2 Tables that might be useful to answering this question. Dr. Lambertsen invited the Navy to further examine these data.

Experimental studies

At least two types of experimental studies could be conducted: studies that examine the effect of thermal factors on DCS risk; and studies that examine mechanisms of thermal effects on DCS risk. A study could be designed to definitively answer whether or not hot water suits increase the risk of DCS. The study could seek to reproduce the dive profile and water temperature conditions of the Flight 800 salvage operation. A possible study matrix would have

one group of divers warmed during the entire dive; a second group would be cold on the bottom and warm during decompression; the third group would be kept cold throughout the dive; and a fourth group would be warm on the bottom and cold during decompression. Decompression time would be held constant. Because the magnitude of the effect is unknown and may be small, several hundred dives would be required to determine if there is any significant difference in risk.

Mechanistic studies could be designed in humans and animals to look at blood flow, gas solubility, and other hypotheses. Such studies could provide valuable information not only for the effect of thermal conditions on DCS, but the broader question of DCS pathophysiology. As such they would provide a useful focus for a biomedical research program.

Conclusion

After reviewing original human studies examining a link between thermal factors and DCS risk we conclude that there is weak support for an effect, but the effect is probably small. Divers should be kept cool on the bottom, but not to the extent that longer bottom times are needed to complete the job, and warm during decompression. If the bottom phase is warm (*e.g.*, wearing hot water suits) consider extra decompression (*e.g.*, apply the next longer schedule). Further analysis of existing thermal and DCS data might be fruitful in strengthening our confidence in a causal relationship. A precise estimate of the magnitude of any thermal effect would require a major human dive trial. Examination of hypothesized mechanisms of action might provide insight into the broader question of DCS pathophysiology.

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